Applied Psychophysiology and Biofeedback, Vol. 29, No. 1, March 2004 (© 2004)

Physiological Responsiveness of Motor Vehicle Accident Survivors With Chronic Posttraumatic Stress Disoder

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This study sought to replicate past research that has shown differences in physiological responsiveness among survivors of motor vehicle accidents (MVAs) with posttraumatic stress disorder (PTSD) and those survivors who do not develop this disorder. Such physiological differences have been found specifically with heart rate (HR) reactivity. This study also attempts to account for differences among those survivors with PTSD who do respond physiologically in laboratory situations and those who do not show a physiological response when presented with audiotaped descriptions of their accidents. Results replicated the significant differences in HR reactivity between diagnostic groups with chronic PTSD versus those with subsyndromal PTSD and non-PTSD. Variables related to the severity of the diagnosis and trauma were found to discriminate between physiological responders and nonresponders with chronic PTSD.

KEY WORDS: stress disorders; posttraumatic; psychophysiology; accidents; traffic.

One symptom of posttraumatic stress disorder (PTSD) that has been the focus of much research is physiological responses to cues reminiscent of the trauma (American Psychiatric Association, 1994). A fairly large body of research has developed around this symptom beginning with the research of Dobbs and Wilson (1960), who examined this phenomenon in World War II and Korean War veterans. There has been work with both civilian and war-traumatized populations showing that trauma survivors with PTSD display higher rates of physiological reactivity to cues reminiscent of their trauma than do trauma survivors without PTSD or control subjects. For an exhaustive review, see Blanchard and Buckley (1998).

One interesting phenomenon in this literature is the consistent finding that there are differences in physiological reactivity to these cues even among trauma survivors exhibiting PTSD. A portion of survivors with PTSD fails to show this physiological reactivity. An important question is why people with PTSD differ in the severity of this symptom. Does

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1090-0586/04/0300-0051/0 © 2004 Plenum Publishing Corporation

this difference tell us something about the truth of the diagnosis or severity of the disorder, or is the difference merely a reflection of individual differences in physiological reactivity? Individual differences in the severity of this symptom may prove useful for determining treatment response. Determining what leads to physiological reactivity in trauma survivors with PTSD is also important, because prolonged high-level reactivity could put people at risk for certain physical conditions (Blanchard, 1990) or exacerbate preexisting physical conditions.

Although the literature involving physiological responses in trauma survivors with PTSD is quite extensive, the literature examining this response in motor vehicle accident (MVA) trauma survivors is more limited. Thus far, only two studies have examined this relationship in an MVA population. In the first, Blanchard, Hickling, Taylor, Loos, and Gerardi (1994) examined the physiological measures of heart rate (HR), systolic and diastolic blood pressures (SBP and DBP), frontal electromyogram (EMG), and electrodermal activity (EDA). The sample included MVA survivors with PTSD, with subsyndromal diagnoses of PTSD, without PTSD, and a non-MVA control group. The sample was assessed 1–4 months posttrauma. Both idiosyncratic audiotapes of each participant's accident and a standardized videotape containing generic scenes from MVAs were used to determine if different modes of stimulus presentation would result in different physiological reactivity. The results of this study indicated that HR response to one of the idiosyncratic audiotapes showed the best discrimination with the MVA-PTSD group exhibiting a greater HR response than each of the other three groups. Finally, there was no basal difference between the PTSD group and the other comparison groups, as has been found in war veteran populations (Blanchard, 1990). Another relevant finding was that a HR change of +2.0 BPM to the audiotape significantly separated the PTSD group from the non-PTSD group and the control group.

Blanchard, Hickling, Buckley, et al. (1996) replicated the results of Blanchard et al. (1994) on a different sample using the same four comparison groups. The physiological responses measured were HR, SBP, DBP, and frontal EMG. Again, participants were presented with two idiosyncratic audiotapes and a standardized videotape. Blanchard, Hickling, Buckley et al. (1996) also found no basal differences on any of the variables among the four groups. They again found that HR response to one of the audiotapes discriminated the MVA-PTSD group from the other three comparison groups. Also, a HR change of +2.0 BPM was found to be a reliable discriminator between the groups as in Blanchard et al. (1994).

This paper addresses these issues with a new, treatment-seeking cohort of MVA survivors: Part 1 seeks to replicate differences in parameters of physiological reactivity that were identified in the previous studies (Blanchard et al., 1994; Blanchard, Hickling, Buckely, et al. 1996) to discriminate cases of acute PTSD (1–4 months post-MVA) from cases without PTSD in a new, more chronic population of MVA survivors who were on average 12.4 months postaccident; Part 2 attempts to explain differences in physiology reactivity among those MVA survivors with chronic PTSD.

PART 1

Method

Assessment

In this study, survivors of MVAs were solicited for possible psychological treatment through newspaper, television, and word-of-mouth referrals. The survivors must have been at least 6 months but not more than 2 years postaccident. After going through a brief phone screening procedure that utilized the PTSD Check List (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) to screen for PTSD symptoms, participants were given an appointment for an extensive assessment. Written informed consent was obtained first at the initial assessment. This assessment included an interview developed by Blanchard and Hickling (1997) to elicit details of the MVA, the Clinician Administered PTSD Scale (CAPS; Blake et al., 1997), LIFE Base (Keller et al., 1987), Structured Clinical Interview for the *DSM-IV* Axis I disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996), and Structured Clinical Interview for *DSM-IV* Axis II personality disorders (SCID-II; First, Spitzer, Gibbon, Williams, & Benjamin, 1996), and an assessment of previous trauma.

Three self-report psychological tests were also utilized including the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the State-Trait Anxiety Inventory (STAI) scored for both state anxiety and trait anxiety (Spielberger, Gorsuch, & Lushene, 1970), and the PCL (Weathers et al., 1993).

Participants were then scheduled for the physiological assessment that replicated the assessments described in Blanchard et al. (1994) and Blanchard, Hickling, Buckley, et al. (1996). The physiological assessment lasted for approximately 36 min and measured HR, SBP, and DBP with a Kritikon Dinamapp, and electrodermal activity (skin resistance level) with a Grass Polygraph. These measures were taken every 60 s throughout the assessment. The phases of the assessment were as follows: baseline 1 (5 min), mental arithmetic (3 min), baseline 2 (5 min), Audio 1 (3 min), baseline 3 (5 min), Audio 2 (3 min), baseline 4 (5 min), relaxation (2 min), baseline 5 (5 min). Mental arithmetic was the neutral stressor consisting of instructing the participant to count backward from 100 by sevens. The relaxation phase consisted of scripted deep-breathing instructions combined with a relaxing beach or wooded scene, depending on the participant's preference. The audiotaped descriptions of the MVAs were idiosyncratic for each participant and were made by the assessor who conducted the initial interviews. These scenes included vivid imagery details of the accident as well as individual thoughts and emotions elicited from the participants during the interview process. See Blanchard and Hickling (1997) for more details concerning the content of the idiosyncratic descriptions. Assessments were conducted by doctoral students in clinical psychology with 1–4 years of experience in conducting the assessments.

Participants

As noted above, all participants were treatment-seeking. One hundred thirty-two participants completed both phases of the assessment. Of these 92 warranted a diagnosis of PTSD on the basis of the CAPS; 23 warranted a subsyndromal diagnosis; and 17 received no diagnosis. subsyndromal PTSD was defined as a participant meeting two of the symptomatic criteria of PTSD according to the CAPS rather than all three required for a full diagnosis. The average age was 40.5. There were no differences among the diagnostic groups on gender, age, or ethnicity. Refer to Table I for demographic characteristics of the diagnostic groups.

Diagnoses of major depression and generalized anxiety disorder were assessed utilizing the SCID-I. The PTSD diagnostic groups differed on the presence of major depression, with the PTSD group being more likely to have comorbid major depression than the subsyndromal or non-PTSD groups (Cramer's $V = .4$, $p < .001$). There were no differences between the

Table I. Diagnostic and Demographic Characteristics of MVA Survivors by Diagnostic Group

Characteristics	PTSD	Subsyndromal	Non-PTSD	Total
N	92	23	17	132
%	70	17	13	100
Gender (male/female)				
N	23/69	9/14	3/14	35/97
% male	25	39	18	27
Ethnicity (White/non-White)				
N	84/8	19/4	15/2	118/14
% White	91	83	88	89
Age				
M	39.67	41.91	43.35	40.54
SD	11.14	13.22	12.58	11.69
Range	20–80	20–70	21–65	20–80
Months since MVA				
M	12.77	12.28	10.73	12.42
SD	8.83	7.72	6.98	8.40
CAPS score				
M	72.36	36.30	16.82	58.92
SD	20.42	11.40	7.70	27.66

diagnostic groups on generalized anxiety disorder (Cramer's $V = .09$, $p = .56$). Refer to Table II for number and percentage of participants in each PTSD group experiencing major depression or generalized anxiety disorder.

Reliability of the CAPS diagnosis was established by having 49 audiotapes of the CAPS portion of the interview rescored by graduate students blinded to diagnosis. Kappa for diagnostic agreement was 0.789, $p < .001$.

This study focused only on HR reactivity scores because this was the only measure found to differentiate between MVA survivors with and without PTSD in the studies by Blanchard et al. (1994) and Blanchard, Hickling, Buckley, et al. (1996). HR reactivity scores for each stressor were calculated by subtracting the mean of the baseline before each stressor phase from the highest HR score obtained during each stressor phase. For example, the HR reactivity score for Audio 1 was computed by subtracting the mean HR score, averaged from the five HRs measured during baseline 2, from the highest HR score of the three measured during the Audio 1 phase. It should be noted that of the 92 participants with PTSD, 3 withdrew from the physiological assessment after the mental arithmetic stressor and 1 withdrew after the presentation of Audio 1. This resulted in 89 with reactivity scores to Audio 1 and 88 with reactivity scores to Audio 2.

Table II. Major Depression and Generalized Anxiety Disorder by CAPS PTSD Diagnostic Category

	PTSD	Subsyndromal	Non-PTSD
Major depression			
Present	52 (57%)	5 (22%)	1 (6%)
Absent	40 (43%)	18 (78%)	16 (94%)
Generalized anxiety disorder			
Present	24 (26%)	4 (17%)	3 (18%)
Absent	68 (74%)	19 (83%)	14 (82%)

Note. Percentage within parenthesis represents the percentage of participants in each cell.

Table III. Mean Physiological Reactivity Scores for Heart Rate for All Diagnostic Groups for All three Stressors

Condition	PTSD		Subsyndromal		Non-PTSD	
	M	*SD*	*M*	*SD*	*M*	*SD*
Mental arithmetic	9.2_a	6.5	9.7_a	7.3	9.3_a	4.9
Audiotape 1	7.0_a	7.0	1.4_b	4.0	3.9_b	4.0
Audiotape 2	5.2_a	4.4	3.5_a	5.0	3.3_a	3.7

Note. Means within a row, which share a subscript, are not different at the .05 level.

Results

A repeated measures ANOVA was conducted to examine the differences between the three diagnostic groups on the three within-participants HR reactivity variables for mental arithmetic, Audio 1, and Audio 2. The main effect of diagnostic group was significant ($F = 3.27$, $df = 2, 124$, $p = .041$) with the PTSD group ($M = 6.94$) and the non-PTSD group ($M = 5.48$) showing an overall higher mean on the three reactivity scores combined than that of the subsyndromal group ($M = 4.85$). The main effect of stressor was also significant ($F = 30.81$, $df = 2, 248$, $p < .001$), with reactivity to mental arithmetic ($M = 9.37$) being higher than both reactivity to Audio 1 ($M = 3.92$) and Audio 2 ($M = 2.98$) that did not differ from one another. Importantly the interaction of stressor by diagnostic group was also significant ($F = 4.405$, $df = 4, 248$, $p = .002$).[6]

A posthoc analysis of covariance was conducted to interpret the interaction. HR scores as measured at baseline 1 were used as the covariate to control for individual differences in HR. This analysis revealed that the differences between the diagnostic groups were significant only on Audio 1 reactivity scores ($F = 8.86$, $df = 2, 123$, $p < .001$) with the PTSD group showing significantly higher reactivity to Audio 1 than either the subsyndromal or non-PTSD diagnostic groups, which were not different from each other. There were no differences between the groups on reactivity to mental arithmetic ($F = 0.27$, $df = 2, 123$, $p = .767$) or Audio 2 ($F = 2.33$, $df = 2, 123$, $p = .101$). Table III displays the means for the diagnostic groups for each of the three physiological reactivity scores.

A separate analysis of variance was conducted on the diagnostic groups for the baseline measure of HR. This revealed no significant differences between the PTSD ($m = 73.0$), subsyndromal ($m = 70.9$), and the non-PTSD ($m = 77.0$) groups on this measure ($F = 1.78$, $df = 2, 131$, $p = .173$).

Individual Responses

On the basis of the results of the previous research (Blanchard et al., 1994; Blanchard, Hickling, Buckley, et al., 1996), a HR response to Audio 1 of +2 BPM or greater was utilized to differentiate physiological responders from nonresponders in the present study. On the basis of this cutoff, 71 (77%) of the PTSD participants are responders and 18 (23%) are nonresponders. The next study examined differences between these responders and nonresponders.

[6]As in earlier studies of MVA survivors, BP and EDA did not show statistically significant differential reactivity among the PTSD, sub- and non-PTSD groups.

Discussion

Previous research on the reliability of HR reactivity to idiosyncratic audiotaped descriptions of MVAs to discriminate MVA survivors with PTSD from those with a subsyndromal diagnosis and no diagnosis was replicated in a sample of MVA survivors 6 months to 2 years posttrauma. Thus, this methodology works as well with chronic MVA-PTSD as it did with acute PTSD. Consistent with previous research (Blanchard et al., 1994; Blanchard, Hickling, Buckley, et al., 1996), there were no basal HR differences between the diagnostic groups. These results strengthen the diagnostic utility of HR reactivity to idiosyncratic audiotaped descriptions in MVA survivors with PTSD. Also consistent with the previous research, there was a failure of a noticeable portion (23%) of MVA survivors with PTSD to show an HR response of greater than 2 BPM to such descriptions. In Part 2, differences within the PTSD diagnostic groups between those survivors who displayed HR reactivity to Audio 1 versus those who do not show such reactivity were examined.

PART 2

Studies have consistently found a certain portion of trauma survivors with PTSD who do not exhibit physiological responsiveness in laboratory situations. It can be asked if there are psychological differences between those who respond and those who do not respond physiologically. Three studies thus far have examined predictors of physiological responsiveness versus nonresponsiveness. Blanchard, Kolb, Taylor, and Wittrock (1989) first examined such differences in a sample of Vietnam veterans. Keane et al. (1998) also examined such differences in a sample of Vietnam veterans, and Blanchard, Hickling, Buckley, et al. (1996) examined such differences in a sample of acute MVA survivors.

Blanchard et al. (1989) found a 25% nonresponder rate and examined measures of anxiety and depression as measured by the Minnesota Multiphasic Personality Inventory, BDI, STAI, Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), and Hamilton Anxiety Rating Scale (HARS; Hamilton, 1959). They found no differences between responders and nonresponders on any of these measures.

Blanchard et al. (1996) examined the differences between responders and nonresponders in a sample of 61 MVA survivors with acute PTSD. They separated groups into responders and nonresponders on the basis of HR response of +2.0 BPM to Audio 1. This resulted in 40 responders and 21 nonresponders, an approximately 34% nonresponder rate. They compared responders and nonresponders with a series of one-tailed *t* tests on variables of anxiety (STAI-state and STAI-trait), depression (BDI), total PTSD symptomatology (total CAPS score), and response to symptom 17 (physical reactivity to cues reminiscent of the trauma, *DSM-III-R*; CAPS item 17). The only variable significantly different between these groups was state anxiety with responders being higher on this measure.

In the largest study to address this topic, Keane et al. (1998) diagnosed 1,168 Vietnam combat veterans who were seeking VA services with the SCID (Structured Clinical Interview for *DSM-III-R*; Spitzer, Williams, Gibbon, & First, 1989). Of the 1,168 veterans, 773 had a current diagnosis of PTSD, 181 met lifetime criteria for PTSD, and 369 never met diagnostic criteria for PTSD. The diagnostic and psychometric assessment included the following: The War Stress Interview (Rosenbeck & Fontana, 1989), SCID, SCID-II modules for borderline and antisocial personality disorders, Minnesota Multiphasic Personality Inventory

(MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1990), The Keane PTSD scale (PK; Keane, Malloy, & Fairbank, 1984), the Mississippi Scale for Combat-related PTSD (Keane, Caddell, & Taylor, 1988), and the Laufer-Parsons Inventory (LPI; Laufer, Yager, Frey-Wouters, & Donnellan, 1981), which assesses war-related guilt.

The psychophysiological assessment stressors included mental-arithmetic, a neutral audiovisual stressor, general combat-audiovisual stressors, and idiographic imagery audio stressors. Physiological measures included HR, skin conductance (SC), EMG, SBP, and DBP. Responders and non-responders were identified on the basis of a logistic regression equation predicting diagnostic status based solely on physiological variables. The 773 veterans with a current diagnosis of PTSD were ranked according to the logistic probability scores from this equation. The 120 PTSD participants with the highest probability scores were classified as responders, whereas the 120 PTSD participants with the lowest probability scores were classified as nonresponders.

Variables were then selected to differentiate between responders and nonresponders. Variables were selected from the areas of social, occupational, legal, military, trauma-related, general psychopathology, and personality. They found two general groups of variables that discriminated between the two groups: severity of PTSD and guilt and depression. For severity of PTSD, the physiological responders showed higher scores on the Mississippi Scale, higher clinician severity ratings of PTSD, less nightly sleep, and less sense of control during idiographic trauma scripts. Responders also experienced more combat exposure, more distress upon exposure to trauma reminders, and were more likely to receive a service-connected disability. These variables suggest that the responders in this sample appear to have more severe symptoms of PTSD, more exposure to trauma, and poorer functioning compared to nonresponders (Keane et al., 1998). With regard to the group of variables indicating guilt and depression, there were three primary differentiating variables: the LPI, the Depression Scale from the MMPI-2, and item 12 from the CAPS (sense of foreshortened future). Selected items from the LPI that show feelings of guilt and depression are "I should have died in the war; I get upset because I feel a buddy or comrade got killed because of something I did or did not do" (Keane et al., 1998). These variables suggest that negative affect plays a role in physiological reactivity.

This study examined variables that have been previously reported to differentiate physiological responders and nonresponders using the present sample of MVA survivors with PTSD as well as tested for new variables that may prove useful in making this discrimination.

Method

Complete psychological test and psychosocial interview data were obtained for 89 of those 92 MVA survivors with chronic PTSD described above. They were divided into responder and nonresponder groups on the basis of a HR reactivity cutoff of +2 BPM reactivity to Audio 1. This resulted in 18 (20%) nonresponders and 71 (80%) responders. These groups were further differentiated by removing those participants with reactivity scores between +2 and +3 BPM. This was done to achieve some degree of separation between the two groups. This resulted in 10 cases being removed, which left 18 (23%) nonresponders and 61 (77%) responders. Table IV displays the demographic information for these two groups. There were no differences on demographic variables between these two groups.

Table IV. Diagnostic and Demographic Characteristics of Physiological Responders and Nonresponders Among Those With PTSD

Characteristics	Responder	Nonresponder	Total
n	61	18	79
% of total	77	23	
Gender (male/female)			
N	18/43	3/15	21/58
% female	70	83	73
Ethnicity (White/non-White)			
n	56/5	17/1	73/6
% White	92	94	92
Age			
M	39.08	38.44	94
SD	11.47	10.37	11.16
Range	20–80	21–54	20–80
Months since MVA			
M	13.3	11.1	12.8
SD	9	8.3	8.9
Total CAPS score			
M	74.2	67.2	72.6
SD	21.1	20.3	21

Because there have been no findings suggesting clear differences between physiological responders and nonresponders on psychosocial and trauma-related variables, a two-step data analytic approach was undertaken. First, variables hypothesized to be related to physiological reactivity were correlated with HR reactivity scores to Audio 1. Then variables significantly correlated with Audio 1 HR reactivity were entered into a stepwise logistic regression equation predicting responder versus nonresponder status.

Variables were selected for the correlation analysis based on previous research. Variables were selected in an attempt to replicate those examined by Blanchard et al. (1996) including state anxiety, trait anxiety, total BDI score, total CAPS score, and participantive report of physiological reactivity (CAPS item 5). Four of Keane et al.'s variables were duplicated exactly and were also included in the correlation (Keane et al., 1998): clinician PTSD severity rating, nightly sleep (CAP item 13), emotional distress upon exposure to trauma reminders (CAP item 4), and sense of foreshortened future (CAP item 12). Variables of number of days in hospital, number of days missed from work because of the accident, and rating of fear at the time of the accident were included in the correlation analysis to relate to Keane et al.'s variables of severity of PTSD. Finally, self-reported symptom cluster scores of PTSD as measured by the PCL were included in the correlation analysis to examine the relationship between self-reported severity of symptoms and physiological reactivity.

Chi-square analyses were conducted for two dichotomous variables. Litigation status was included based on previous work done by Blanchard, Hickling, Taylor, et al. (1996), which has shown litigation status to be related to the development of PTSD. Acute stress disorder in the month following the accident was included on the basis of the suggestion that depersonalization and derealization may play a role in the development of PTSD (Ehlers, Mayou, & Bryant, 1998). Acute stress disorder was retrospectively diagnosed based on participants' reports of their reactions in the month immediately following the MVA.

Table V. Correlation of Continuous Variables With Heart Rate Reactivity to Audio 1

	Heart rate reactivity to Audio 1
BDI	−.072
State anxiety	.028
Trait anxiety	−.170
PCL total score	.124
PCL Cluster B	.213*
PCL Cluster C	.113
PCL Cluster D	.099
CAPS total score	.182
CAPS 4	.111
CAPS 5	.106
CAPS 12	−.083
CAPS 13	.109
Clinician severity rating	.115
Days missed work	.268*
Days hospitalized	.316**
Fear rating at time of MVA	.182

*$p < .05$. **$p < .01$.

Results

Identification of Individual Predictors

Table V presents the correlations between all of our rationally selected possible predictors and HR reactivity to Audio 1. Of all the variables examined, number of days missed from work, number of days hospitalized, and PCL Cluster B scores were significantly related to HR reactivity scores.

Litigation status was not related to responder status ($\chi^2 = 0.277$, $df = 1$, 79, $p = .599$). Acute stress disorder was also not related to responder status ($\chi^2 = 0.008$, $df = 1$, 79, $p = .928$).

Logistic Regression

Three variables: number of days hospitalized, number of days missed from work, and PCL Cluster B total score were entered into a logistic regression equation using the Forward: LR method. Two of the variables combined to correctly classify 81% of the cases compared to 77.2% correctly classified by predicting everyone to be a responder. These were PCL Cluster B scores and number of days missed from work because of the accident. The improvement in fit relative to the null model as reflected by deviance statistics was

Table VI. Logistic Regression Analysis With Significant Variables

Variable	*B*	*SE*	Wald[a]	*df*	Significance	% Correctly identified
PCL Cluster B score	0.1465	0.0629	5.4301	1	.0198	78.48
Number of days missed from work	0.0061	0.0031	3.7014	1	.0544	81.01
Constant	−1.5606	1.0437	2.2359	1	.1348	

[a]The Wald statistic has a chi-square distribution and indicates whether the *B* coefficient for the predictor is significantly different from zero.

statistically significant ($\chi^2 = 10.88$, $df = 2$, $p < .01$; see Table VI. PCL Cluster B scores being in the model is consistent with responders showing greater severity of PTSD than nonresponders. Number of days missed from work because of the accident is consistent with the idea that more severe traumas may lead to greater reactivity.

Discussion

These results can be viewed to some extent as consistent with those obtained by Keane et al. (1998) in that they suggest that trauma survivors with more severe PTSD symptoms are more likely to be physiological responders than those with less severe PTSD symptoms. However, when the studies examining this difference are viewed as a whole there emerges no clear difference between responders and nonresponders. It is not clear why only certain symptoms emerge as predictors and not others. For instance, the failure of participantive self-report of physiological reactivity to predict objective measures of such raises the possibility that people are unable to accurately report their experience of internal physiological states. Another explanation for the consistent finding of differences in physiological reactivity among trauma survivors with PTSD is that those people who respond physiologically to cues reminiscent of their trauma are more prone to be reactive physiologically in general. If this were the case, we would expect to find that responders exhibited greater reactivity to the neutral stressor, mental arithmetic, which was not the case in this study. The mean HR reactivity for mental arithmetic for responders was 9.9 BPM, the mean reactivity for non-responders was 7.8 BPM ($F = 1.45$, $df = 1, 78$, $p = .232$).

CONCLUSION

The results from the first study showed that HR reactivity to cues reminiscent of the trauma proved to be a useful diagnostic tool in discriminating MVA survivors with PTSD diagnosis from those with a subsyndromal and no diagnosis, even for those who are on average 1 year or more posttrauma. However, there remains a proportion of between 20 and 30% of survivors with PTSD who do not demonstrate this reactivity. Part 2 attempted to determine which psychological factors would be predictive of HR reactivity. Three variables did emerge as significant predictors suggesting that survivors with more intense fear reactions at the time of the accident and more severe symptomatology still present at the time of the assessment were more likely to experience physiological reactivity.

Another interesting related line of research is examining people's immediate physiological reactions to a trauma and their relationship to later PTSD status (Blanchard, Hickling, Galovski, & Veazey, 2002; Bryant, Harvey, Guthrie, & Moulds, 2000; Shalev et al., 1998). It will be interesting to see if such immediate physiological measures will help explain later physiological reactivity as do immediate psychological report of fear reactions. A focus on both psychological and physiological variables will hopefully create a clearer picture of determining which PTSD survivors respond physiologically and which ones do not show such responsiveness.

ACKNOWLEDGMENT

This research was founded by a grant from NIMH, MH-48476.

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